ill-planned surgical procedure had been previously attempted. They did not explain how they made their decision.

In the last century, surgeons treated the congenital vascular malformation (CVM) solely through a surgical approach. This "cavalier approach," with limited knowledge of the natural history of these lesions, often resulted in extremely poor outcomes with high morbidity and high rates of recurrence. Dismal outcomes were due to the natural biologic behavior of the CVM lesion. Surgeons later learned that the embryological characteristics of the CVMs play a critical role in its behavior.

All CVM lesions derived from an "earlier" stage of embryogenesis maintain their mesenchymal cell characteristics, such that they proliferate when stimulated by incomplete excision or simple ligation of a feeding artery. These embryonic tissue remnants, classified as "extratruncular" lesions, are often surgically inaccessible and difficult to remove completely. Therefore, only a carefully planned multidisciplinary approach, combining both endovascular therapy and open surgical treatment, is able to deliver a successful outcome, as the authors demonstrated beautifully.

Second, the authors have made a critical decision to limit the extent of the coil embolization therapy and stent grafting, and utilized the endovascular therapy as a means to reduce the risk of massive bleeding during the subsequent surgery. They did not rely solely on an endovascular approach as an independent and permanent treatment. Again, they did not explain their rational of this approach.

Up until a few decades ago, simple ligation/ablation of a feeding artery of an AVM lesion was often performed without knowledge of the characteristics of extratruncular AVM lesions. This maneuver deprives the AVM lesion of its arterial supply only temporarily, which then ultimately stimulates this embryonic tissue remnant with mesenchymal cell characteristics. The nidus of the lesion will soon proliferate and develop new arterial collaterals via "neovascular recruitment," making the condition worse. Therefore, ligation/ablation of the feeding artery as a sole, permanent treatment should no longer be practiced as the authors wisely demonstrated.

Third, the authors made another critical decision to perform ethanol sclerotherapy[3] prior to surgical excision. This was a wise decision. As before, they did not explain clearly their rationale and why they took this approach.

The only way to achieve a curative resection of an extratruncular AVM lesion is to completely destroy the endothelial cells of the nidus in order to prevent its recurrence. Ethanol is the only sclerotherapy agent that has been shown to deliver permanent destruction of the lesion nidus (with mesenchymal cell characteristics). Because this extratruncular lesion is not a well localized one, but rather a diffusely infiltrating lesion, preoperative embolization therapy with N-butyl cyanoacrylate would not have been as good a choice as ethanol sclerotherapy to deliver effective local control. Ethanol sclerotherapy provided extra reassurance of permanent destruction of any potential residual cells present following excision.

The authors have successfully utilized a multidisciplinary approach to the treatment of a high risk AVM lesion,[4] combining both endovascular therapies with open surgical resection, resulting in an excellent outcome. We continue to support and promote this multidisciplinary approach to the AVM.

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Reply

I appreciate your comments about my article[1] entitled "Multidisciplinary approach in the management of a giant arteriovenous malformation."

The only reason I did not explain in detail why I chose the multidisciplinary approach to treat this AVM (endovascular therapies plus open surgical resection) was that I had to write the case using only 350 words (as a Vascular Image). I agree with all your comments, and I also think that the multidisciplinary approach is the best strategy to treat these lesions.

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REFERENCE

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Regarding "Pharmacologic risk factor management in peripheral arterial disease: A vade mecum for vascular surgeons"

The excellent article by Rehring et al[1] suggests specific recommendations for tobacco cessation, blood pressure control, lipid-lowering agents, and antiplatelet therapy in patients with peripheral arterial disease (PAD). A risk factor that perhaps deserves more extensive discussion is the presence of diabetes mellitus (DM) in these patients.

The authors mention that, "there are little data to support that aggressive control of blood glucose levels improves risk of MI, stroke, vascular death, or amputation."[1] The results of an earlier study comparing the severity and outcome of PAD in DM (n = 58) and non-DM (n = 68) patients may be of interest.[2] This study showed that during a follow-up period of 4.47 ± 1.25 years for DM vs 4.52 ± 1.23 years for non-DM patients ($P = .85$), DM patients were five times more likely than non-DM patients to undergo an amputation (41.4% vs 11.5%), with an odds ratio (OR) of 5.4 (95% confidence interval [CI], 2.3-12.9; $P < .0001$) and three times more likely to die (51.7% vs 25.6%), with an OR of 3.1 (95% CI, 1.5-6.4; $P = .002$).[2]

As reported in a Consensus Statement of the American Diabetes Association, "the natural history of PAD in diabetic patients has not specifically been studied longitudinally, but it is known from prospective clinical trials of risk interventions that the cardiovascular event rates in patients with PAD and diabetes are higher than those of their nondiabetic counterparts."[3] This can also be extrapolated from the fact that "PAD is marker of systemic vascular disease involving coronary, cerebral and renal vessels, leading to an elevated risk of events, such as myocardial infarction, stroke, and death."[3]